# Headache Caused by Sinus Disease

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**KEYWORDS**

- Migraine • Sinusitis • Autonomic dysfunction • Trigeminovascular pathway
- Low-dose computed axial tomography • Magnetic resonance imaging

**KEY POINTS**

- Headaches and sinus disease are common reasons to seek medical care; symptoms are similar and may relate to autonomic dysfunction and trigeminovascular pathways.
- Headaches from sinus disease are uncommon; most patients with "sinogenic pain" may actually have migraines or tension-type headaches.
- Imaging for acute rhinosinusitis is often not necessary, unless complications or concerns for serious causes, including facial swelling, orbital proptosis, and cranial nerve palsies.
- Sinus radiographs are often inaccurate; multiplanar computed tomography offers advantages of improved bony detail and can be done with low-dose protocols.
- MR imaging may be useful for complex sinus disease, distinguishing polyps, obstructive masses from inspissated secretions and fluid, infraorbital, or intracranial involvement.

## INTRODUCTION

Rhinosinusitis is a common complaint present in 16% of the US population with annual economic burdens estimated at $22 billion.[1] Headaches are also extremely common, affecting 30% to 78% of the population, with US cost estimates of $100 million per million inhabitants per year.[2,3] These 2 conditions are among the top 10 reasons patients seek medical care, especially from otolaryngologists and neurologists.[4] Although patients and clinicians may self-diagnose their symptoms as a "sinus headache" or "rhinogenic headache," there is no true clinical definition for this entity.[5] Many studies have shown that so-called sinus headaches are in fact migraines in up to 88% to 90% of patients.[6,7] The Sinus, Allergy, and Migraine Study found that most patients self-diagnosing themselves or presenting to primary care physicians with a sinus headache from blockage or congestion were actually suffering from migraines.[8] Confounding the issue are the 2013

International Headache Society *International Classification of Headache Disorders* headache categories, which include 11.5 Headache attributed to disorder of the nose or paranasal sinuses, 11.5.1 Headache attributed to acute rhinosinusitis, and 11.5.2 Headache attributed to chronic or recurring rhinosinusitis (**Box 1**).[9] The similar overlapping symptoms of sinusitis and migraine likely occur due to similar anatomic autonomic, trigeminal nerve, vidian nerve, and the trigeminocardiac reflex pathways. This article reviews the anatomy, clinical cases, how imaging plays a role in assessment, and essential key clinical and radiographic findings that separate these entities.

## NORMAL ANATOMY
### Sinuses and Drainage Pathways

The air-filled spaces of the paranasal sinuses are lined with respiratory epithelium with cilia working together to clear secretions. At birth (**Fig. 1**), ethmoid and maxillary sinuses are present and

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**Box 1**
**Classification of headache disorders**

11.5. Headache attributed to disorder of the nose or paranasal sinuses. Previously used term: The term "sinus headache" is outmoded because it has been applied both to primary headaches and headache supposedly attributed to various conditions involving nasal or sinus structures. *Description:* Headache caused by a disorder of the nose and/or paranasal sinuses and associated with other symptoms and/or clinical signs of the disorder.

11.5.2. Headache attributed to chronic or recurring rhinosinusitis

*Description:* Headache caused by a chronic infectious or inflammatory disorder of the paranasal sinuses and associated with other symptoms and/or clinical signs of the disorder. Diagnostic criteria:

A. Any headache fulfilling criterion C

B. Clinical, nasal endoscopic, and/or imaging evidence of current or past infection or other inflammatory process within the paranasal sinuses

C. Evidence of causation demonstrated by at least 2 of the following:

   1. Headache has developed in temporal relation to the onset of chronic rhinosinusitis

   2. Headache waxes and wanes in parallel with the degree of sinus congestion, drainage, and other symptoms of chronic rhinosinusitis

   3. Headache is exacerbated by pressure applied over the paranasal sinuses

   4. In the case of a unilateral rhinosinusitis, headache is localized ipsilateral to it

D. Not better accounted for by another International Classification of Headache Disorders-3 (ICHD-3) diagnosis.

*Comment:* It has been controversial whether or not chronic sinus pathology can produce persistent headache. Recent studies seem to support such causation.

11.5.1. Headache attributed to acute rhinosinusitis

*Description:* Headache caused by acute rhinosinusitis and associated with other symptoms and/or clinical signs of this disorder.

   Diagnostic criteria:

A. Any headache fulfilling criterion C

B. Clinical, nasal endoscopic, and/or imaging evidence of acute rhinosinusitis

C. Evidence of causation demonstrated by at least 2 of the following:

   1. Headache has developed in temporal relation to the onset of the rhinosinusitis

   2. Either or both of the following:

      a. Headache has significantly worsened in parallel with worsening of the rhinosinusitis

      b. Headache has significantly improved or resolved in parallel with improvement in or resolution of the rhinosinusitis

   3. Headache is exacerbated by pressure applied over the paranasal sinuses

   4. In the case of a unilateral rhinosinusitis, headache is localized ipsilateral to it

D. Not better accounted for by another ICHD-3 diagnosis.

*Comments:* 1. Migraine and 2. Tension-type headache can be mistaken for 11.5.1 Headache attributed to acute rhinosinusitis because of similarity in location and, in migraines, because of the commonly accompanying nasal autonomic symptoms. The presence or absence of purulent nasal discharge and/or other features diagnostic of acute rhinosinusitis help to differentiate. However, an episode of 1. Migraine may be triggered or exacerbated by nasal or sinus pathology. Pain as a result of pathology in the nasal mucosa or related structures is usually perceived as frontal or facial but may be referred more posteriorly. Finding pathologic changes on imaging of acute rhinosinusitis, correlating with the patient's pain description, is not enough to secure the diagnosis of 11.5.1 Headache attributed to acute rhinosinusitis. Treatment response to local anesthesia is compelling evidence, but may also not be pathognomonic.

*From* Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition (beta version). Cephalalgia 2013;33(9):629–808.
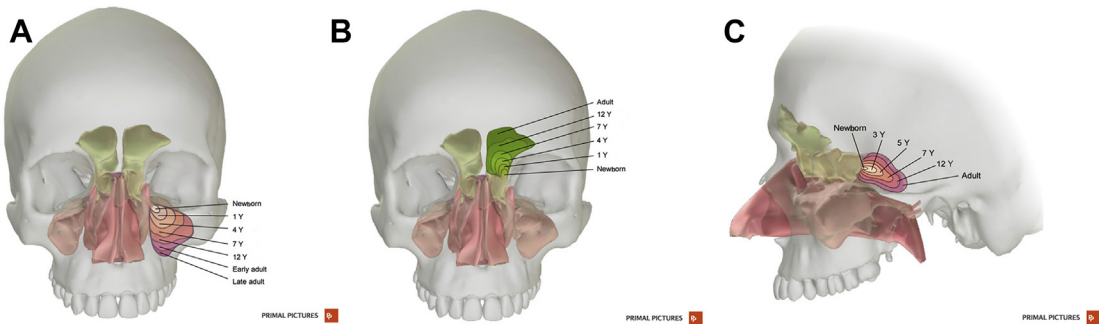
**Fig. 1.** (*A–C*) Sinus development. (*A, B*) Ethmoid and maxillary sinuses are present at birth and sites of infection in pediatric patients; the frontal sinuses arise from anterior ethmoidal air cells at around 6 years old. (*C*) Sphenoid sinus develops from age 2 years and pneumatizes at around age 8 years.

are the 2 major sites for infection in pediatric patients.[10] Sphenoid sinus pneumatization starts at about 9 months and the frontal sinuses at 7 to 8 years of age, with continuous expansion into adolescence. In children, acute rhinosinusitis (ARS) is a clinical diagnosis, and radiographic imaging is not indicated unless concerns for complications or surgical planning.[11] Imaging in patients with uncomplicated ARS is not proven to be useful, in that up to 80% of uncomplicated AR patients may have abnormal radiographic finding.[12]

Sinus anatomy is shown in **Fig. 2**. The superior frontal sinuses may be variable in size: 4% of the population may be hypoplastic and 5% to 8% of the population may be aplastic.[13,14] Frontal sinuses drain via the frontal recess, bordered by the fovea ethmoidalis, and the roof of the ethmoid air cells superiorly. At the anterior frontal recess margin is the agger nasi air cell, the most anterior ethmoid air cell. At the posterior frontal recess margin is the ethmoid bulla, located posterior superior to the hiatus semilunaris. The lateral wall of the olfactory fossa forms the medial frontal recess margin and the lamina papyracea the lateral margin; the opening of the frontal recess may vary depending on the attachment of the UP opening into either the infundibulum or the middle meatus.[15]

The nasolacrimal duct (NLD) (**Fig. 2**A) extends inferiorly from the ocular surface to the nasal cavity inferior meatus and contains an upper and lower canaliculus. The NLD walls are composed of helical connective tissue with a unique combination of microvilli, seromucous glands, lymphocytes, and macrophages[16] (**Fig. 2**A). An easy mnemonic to remember sinus drainage is anterior inferior–posterior superior. The most anterior structures, that is, the NLDs, drain to the inferior meatus; just behind them going posteriorly are the frontal recess, and anterior ostiomeatal complex draining the maxillary sinuses and anterior ethmoid air cells

into the middle meatus, the posterior ethmoid air cells, and the sphenoid sinus. The most posterior sinuses drain via the sphenoethmoidal recess into the superior meatus.

The ostiomeatal complex should be assessed for patency and is formed by the important bony uncinate process (UP) (**Fig. 2**B); this bone is what may be removed in functional endoscopic sinus surgery to visualize the maxillary sinus opening or ostium. Therefore, the radiologist and surgeon need to assess preoperatively that it is not attached or atelectatic to the orbital lamina papyracea.[17] The UP forms the medial maxillary infundibulum margin; the infundibulum is marked by pink arrows in **Fig. 2**B. The UP free edge superiorly forms the inferior hiatus semilunaris margin; this drains to the medial meatus and may vary anatomically, as in **Fig. 2**B, with a pneumatized uncinate tip.

## SINUS ANATOMY: NEURAL AND VASCULATURE

Many neural pathways involving nociception, parasympathetic, and sympathetic nerves are involved with the paranasal sinuses. The nasal cavity takes inhaled air and filters, warms, humidifies the inhaled air, and is critical for perceiving noxious odors and stimuli. How smell works is yet to be completely elucidated, although CN I, the olfactory nerve, is critical, and CN V, the trigeminal nerve, also plays a role.

### Cranial Nerve I: Olfactory Nerve

CN I, the unique olfactory nerve composed of unmyelinated axons with unique glia cells, has marked plasticity, allowing continual replacement of axons and remodeling in the central nervous system. However, CN I can also give viruses direct access intracranially. CN I joins axons forming fascicles "fila olfactoria," through the ethmoid lamina cribosa (cribriform plate
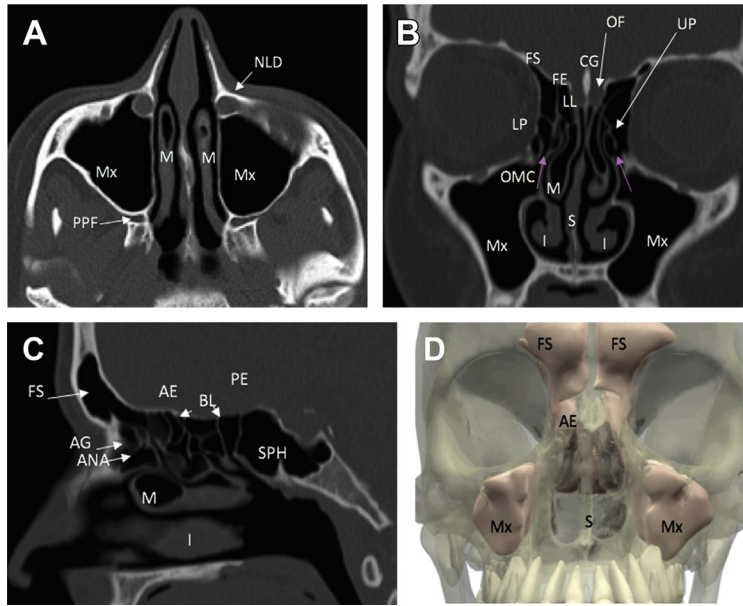
**Fig. 2.** (*A–D*) Bone window sinus CT normal paranasal sinuses axial plane (*A*), coronal plane (*B*), and sagittal plane (*C*). Sinus (*D*). AE, anterior ethmoid air cells, drain via ostiomeatal complex infundibulum to middle meatus, air space around middle turbinate; AG, Agger nasi (*Agger*, Latin for mound, pile, rampart; *Nasi*, nose), lateral nasal cavity ridge formed mucosa over maxilla ethmoidal crest; *ANA*, Agger nasi air cell, 90% of patients in lacrimal bone below AG, enlargement may obstruct frontal recess; BL, basal lamella (Latin *lamella*, small plate/flake), separates anterior and posterior ethmoid sinus, third basal lamella laterally attaches to lamina papyracea; CG, crista galli (Latin rooster crest) superior ethmoid bone above cribriform plate, attachment for anterior falx cerebri; FE, fovea ethmoidalis (Latin *fovea*, pit, depression) from the frontal bone forms ethmoid roof separating from anterior cranial fossa; FS, frontal sinus, drain frontonasal duct to anterior middle meatus; I, inferior turbinate (Latin turbinate inverted cone, scroll shaped, top), largest turbinate, deflects, humidifies, heats, filters air space or meatus; LL, lateral lamella, thinnest bone in the body, vertical lamella, joins inferior fovea ethmoidalis, variable height (Keros classification) affects olfactory fossa depth; LP, lamina papyracea (Latin *papyracea*, paper thin) from ethmoid bone, forms medial orbital wall, lateral ethmoid sinus; M, middle turbinate (also known as nasal concha, inferior ethmoidal turbinate, Latin *concha*, like seashell) when air filled, called concha bullosa; Mx, maxillary sinus, largest sinus drains to middle meatus; NLD, carries tears from eye to nasal cavity into inferior meatus; OF, olfactory fossa, contains olfactory nerve and bulb; OMC, ostiomeatal complex, pink arrows show the maxillary infundibulum, key drainage for the frontal, anterior ethmoid and maxillary sinuses, contains frontal recess, maxillary sinus ostium, ethmoidal infundibulum, hiatus semilunaris, middle meatus; PE, posterior ethmoid sinus, drains via sphenoethmoidal recess to the superior meatus with sphenoid sinus; S, nasal septum, separates nasal cavity formed by ethmoid perpendicular plate, vomer, crest of maxillary bone, crest of palatine bone, septal nasal cartilage, where the vomer and perpendicular plate of the ethmoid fuse there may be a bony spur; SPH, sphenoid sinus variable in shape, drains via sphenoethmoidal recess (or posterior ostiomeatal complex) to superior meatus; UP (Latin *uncinate*, hooked), note both of these are pneumatized, a normal anatomic variant. The UP arises from the lateral wall ethmoid cavity, a key component of the ostiomeatal complex, arises from ethmoid bone, superior edge forms an inferior margin of the hiatus semilunaris, and ethmoid bulla forms upper margin.

intracranially with sensory neurons connecting the brain and nasal cavity with no relay). The olfactory bulb circuitry contains olfactory axons and olfactory glomeruli.[18] Each olfactory bulb in the olfactory recess (**Fig. 2**B, **Fig. 3**), lateral to the crista galli, contains layered juxtaglomerular cells, containing periglomerular cells, external tufted cells, superficial short axon cells, mitral cells, and tufted cells. The olfactory pseudostratified columnar neuroepithelium has ciliated olfactory receptors in the nasal vault below the cribriform plate and also extends to the superior nasal septum, superior turbinate, and superior lateral nasal wall in the olfactory cleft. There are 10 to 20 million cell bodies of primary olfactory receptor neurons.[19,20] These "filia olfactoria" nerves traverse the cribriform plate minute 15 to 20 openings and synapse in the olfactory bulb. The nasal cavity neuroepithelium along with olfactory receptor neurons contains microvillar cells, sustentacular cells, horizontal, globose basal cells, and Bowman gland duct
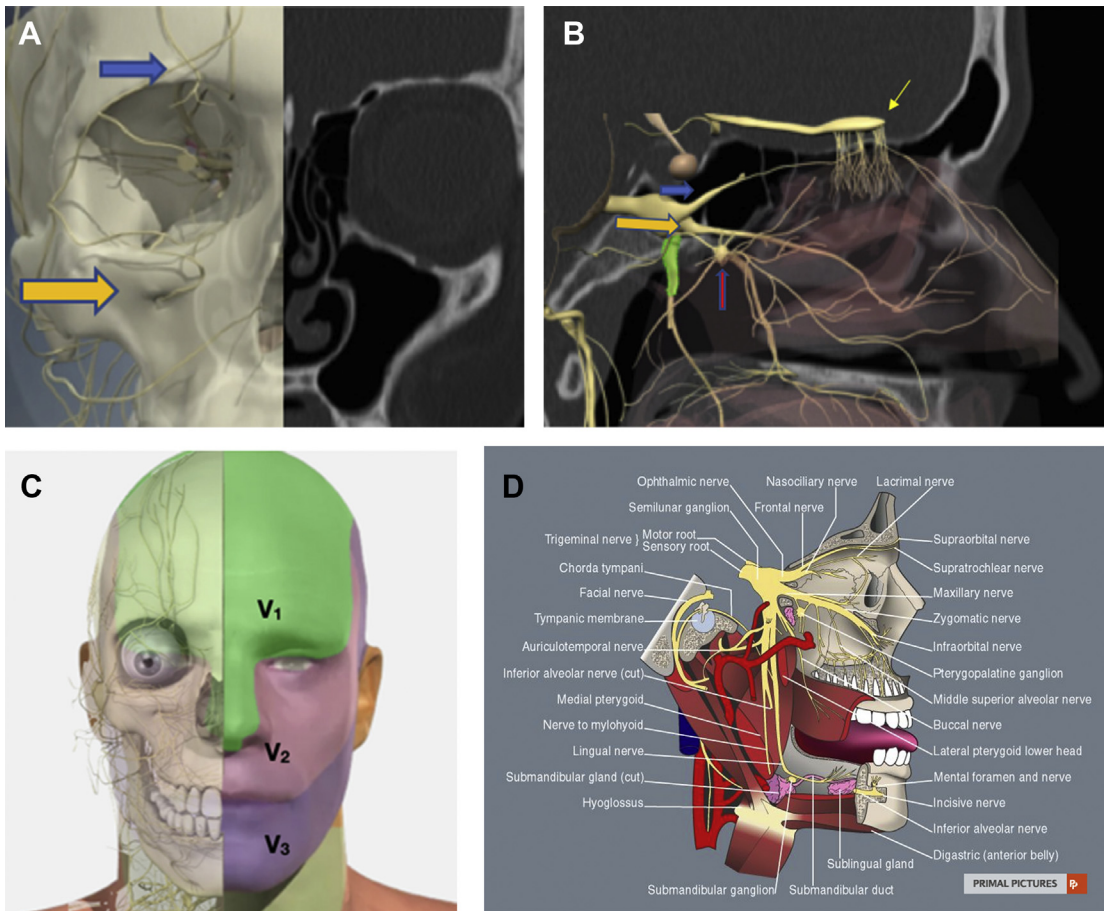
**Fig. 3.** (*A*) Coronal CT bone windows on the right side, with diagrammatic overlay on the left side showing the exit points of CN trigeminal branches. (*B*) Sagittal sinus CT with schematic overlay showing branches of CN I, olfactory nerve with filia olfactoria through the cribriform plate (*yellow arrow*), CN V branches including $V_1$, ophthalmic nerve that exits via the superior orbital fissure, with distal supraorbital branch (*small blue arrow*). $V_2$, maxillary nerve that exits via the foramen rotundum, synapses in the pterygopalatine ganglion (*red arrow*) to the infraorbital fissure and exits via the infraorbital foramen (*orange arrow*); and CN $V_3$, mandibular nerve (marked in green) that exits via the foramen ovale. (*C*) Sensory innervation distribution of the CN V trigeminal branches. (*D*) Detailed labeling in the sagittal plane of CN V trigeminal branches.

cells, which create mucus, allowing smell transduction.[21] Human olfactory neurons regenerate every 3 to 6 months, declining as one ages. Odor is detected by primary order olfactory receptors synapsing with second-order dendrites of mitral and tufted cells in the olfactory bulb glomerus and then is sent to anterior olfactory nucleus, olfactory tubercle, piriform cortex, lateral entorhinal cortex, amygdala cortical nucleus, periamygdaloid cortex, with fibers to the lateral hypothalamus and hippocampus, with smell intrinsically linked to memory.[22]

### Cranial Nerve V: Trigeminal Nerve

The sensory supply of the paranasal sinuses is from $V_1$ ophthalmic and $V_2$ maxillary trigeminal neural branches arising after the nerve exits intracranially via the superior orbital fissure, including as shown in **Fig. 3**.

*CN $V_1$ ophthalmic nerve:* exits intracranially via the superior orbital fissure, branches into 3 nerves: frontal, lacrimal, nasociliary.

> *Frontal nerve:* gives rise to the
>> Supraorbital nerve upper lid, frontalis muscle, scalp and branch to the frontal sinus
>> Supratrochlear nerve: supplies conjunctiva, upper lid forehead
> *Nasociliary nerve* gives rise to
>> *Posterior ethmoid nerve:* Arises before anterior ethmoid nerve, supplies posterior ethmoid and sphenoid sinus

*Anterior ethmoid* nerve: supplies frontal and anterior ethmoid sinus, anterior septum

*Internal nerve:* Lateral and medial branches

> *External nasal nerve:* supplies nasal tip skin

*Infratrochlear nerve:* arises near anterior ethmoidal foramen, runs along medial orbit, exits above medial canthus, supplies lateral nose above medial canthus, medial conjunctiva, and lacrimal apparatus.

Several long ciliary nerves enter glove with ciliary ganglion short ciliary nerves

Supplying cornea, iris, ciliary body, eye

*CN V₂–Maxillary nerve:* Exits via foramen rotundum to the pterygopalatine fossa and pterygopalatine ganglion and then continues via the inferior orbital fissure as the *infraorbital nerve* supplies sensory information midface, cheek, maxillary teeth.

Before foramen rotundum, a middle meningeal dural branch goes through foramen spinosum with the middle meningeal artery. After leaving the foramen rotundum, the nerve synapses in the pterygopalatine fossa give a zygomatic branch that divides to the zygomaticotemporal zygomaticofacial branches.

Anteriorly, the *infraorbital nerve* exits the infraorbital foramen and gives rise to the inferior palpebral, external and internal nasal branches, superior labial nerve medial and lateral branches.

*Nasopalatine nerve* via sphenopalatine foramen: goes incisive fossa hard palate

Superior alveolar nerves: anterior, middle, and posterior branches

Posterior superior nasal nerve: lateral branches superior and middle concha

Medical branches: nasal septum

> *Greater and lesser palatine nerves:* branches perforate palatine bone perpendicular plate for sensation of mucosa over inferior nasal concha, inferior and middle meatus.[22–24]

### Autonomic: Parasympathetic/Sympathetic Innervation

The general visceral efferent parasympathetic fibers arise from the superior salivatory nucleus and dorsal pontine lacrimal nucleus exiting into the cerebellopontine angle cistern via the nervus intermedius. The nervus intermedius exits via the internal auditory canal joining the facial nerve motor root and goes through the geniculate ganglion without synapsing to continue as the greater superficial petrosal nerve (GSPN).[25–28] The GSPN extends to the middle cranial fossa, picking up postganglionic sympathetic fibers from the internal carotid artery deep to the petrosal nerve and forms the vidian nerve (also known as the nerve of the pterygoid canal). The vidian nerve exits via the vidian canal and synapses at the pterygopalatine ganglion with branches that subsequently innervate the minor salivary glands of the nasal cavity, palate, and paranasal sinuses.

### Arterial Supply

The highly vascular nose is the only place where blood flows from lateral to medial; this means the arterial blood flow in the mucosa runs anteriorly opposite the direction of inspired air to warm it, with 3 major blood vessels. The key vessel is the sphenopalatine artery arising from the external carotid artery internal maxillary artery. The sphenopalatine artery divides into a septum internal artery and 2 external branches supplying the mucus membranes of the outer margin, concha, ethmoid, and sphenoid sinuses. Arterial supply also arises from the anterior and posterior ethmoid branches originating from the internal ophthalmic artery. Branches as in **Fig. 4**A, B with awareness and localization of the critical anterior and posterior ethmoid arteries are important to avoid inadvertent trauma to the vessels during functional endoscopic surgery. Vessels arising from the external carotid artery include, as noted above, the sphenopalatine artery, greater palatine, superior labial, and angular arteries. The nasal septum receives blood supply from the anterior and posterior ethmoid arteries superiorly and the sphenopalatine artery at the posterior inferior margin, the greater palatine posteriorly, and the labial artery anteriorly. These terminal vessels converge at the inferior anterior third region of the nasal septum, also known as "Little area" after Dr Little, a portion of the septum at risk for nose bleeds, with a highly vascular supply at the transition point between respiratory and squamous epithelium known as "Kiesslbach plexus," essentially a 1.5-mm$^2$ area of capillary loops; bleeding from either anterior or posterior vessels can result in nosebleeds (epistaxis), one of the most common ear, nose, and throat (ENT) emergencies.

### Venous Drainage

The nasal venous supply lacks valves (**Fig. 4**C, D) and accompanies arterial vessels perforating the maxillary bone from the facial, maxillary, infraorbital, and palatine arteries. The veins drain into the anterior facial vein and with venous plexuses at the interior nasal conchae, inferior meatus, and nasal septum posteriorly into a pterygoid plexus. Veins from the orbital ophthalmic plexus join with ethmoidal veins. Venous drainage can vary going either intracranially, intraorbitally, or both. Upper
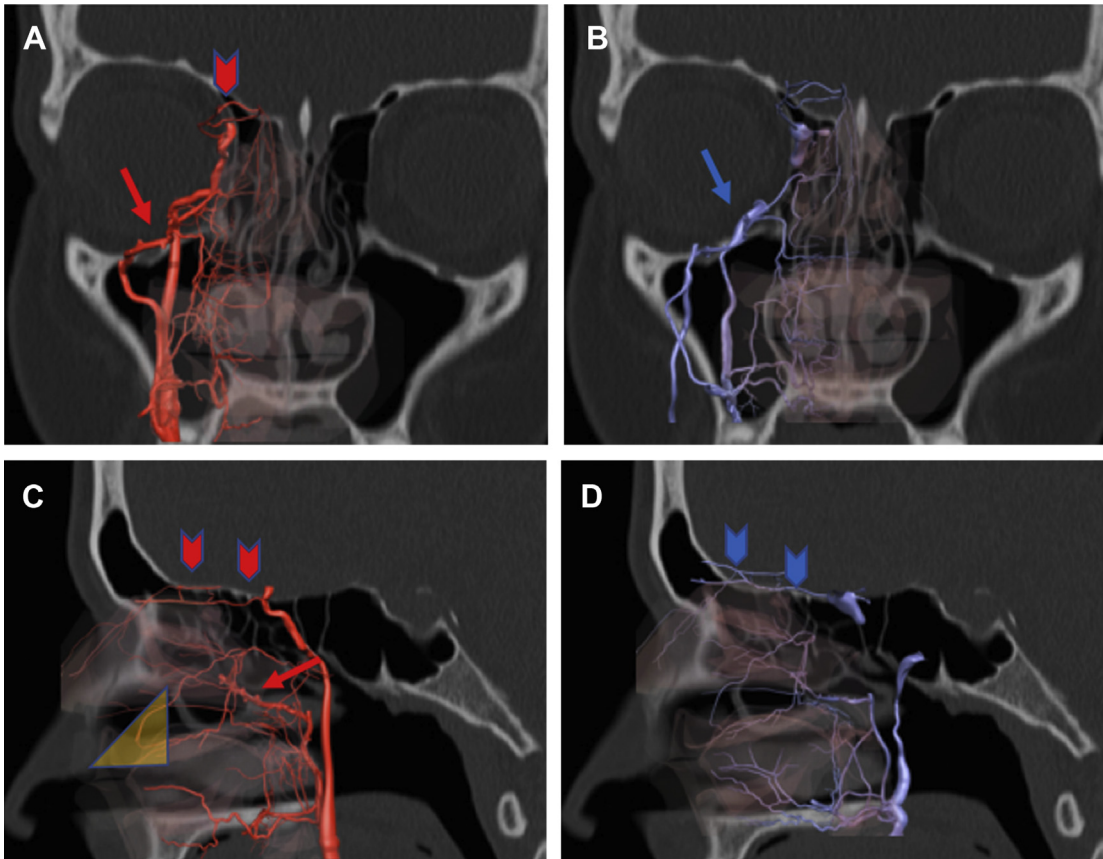
**Fig. 4.** (*A–D*) Bone window sinus CT with arterial supply (*A*) coronal, (*B*) sagittal and venous drainage, (*C*) coronal, (*D*) sagittal. (*A*, *B*) Sphenopalatine artery divides internal artery of the septum external branches (*red arrow*) anterior and posterior ethmoid branches (*red chevrons*) from ICA ophthalmic artery. The nasal septum anterior and posterior ethmoid arteries superiorly and sphenopalatine artery, greater palatine posteriorly, and labial artery anteriorly converge at the inferior anterior third region of the nasal septum. Little triangle in light yellow shows terminal vessels and region at risk for epistaxis. Venous drainage (*C*, *D*), veins follow along arterial pathways and venous drainage may either go to the pterygoid plexus, facial vein, along the orbit or intracranially to the superior sagittal sinus, cavernous, or sphenoparietal sinus.

nasal cavity and frontal sinus veins drain into the interior calvarium via cribriform plate foramina and foramen cecum to the superior longitudinal sinus, superior sagittal sinus, or sphenoparietal sinus.[29] Importantly, pathogens spreading via the veins from the sinuses through the lamina papyracea may be why rhinosinusitis is the predominant cause of pediatric orbital infections.[30–33] The direct connection of veins along the frontal lobes at the orbital margin, via cribriform plate and foramen cecum to the superior sagittal sinus, also allows infections to spread via this route intracranially with risk for venous sinus thrombosis.

## Trigeminocardic Reflex

The blood flow of nasal vessels running opposite to inspired air is controlled via autonomic reflexes.

The trigeminal nerve ophthalmic $V_1$ and maxillary $V_2$ have afferent fibers sending the sensation of the paranasal sinuses to the trigeminal brainstem sensory nuclear complex, which includes the spinal trigeminal nucleus, thalamus, and somatosensory cortex. Autonomic sympathetic stimulation is from nerve fibers arising from the superior cervical ganglion via the deep petrosal nerve branch of the vidian nerve; these fibers join with parasympathetic fibers from the CN VII superior salivatory nucleus that form the GSPN. Together the deep petrosal nerve and GSPN form the vidian nerve synapsing in the sphenopalatine ganglion. Both the trigeminal nerve endings and the parasympathetic nerves end in the basal cells of the nasal epithelium.[34] Sympathetic stimulation in the nasal cavity decreases blood flow and in doing so decongests the nasal venous erectile tissue. [29,35].

Pain in the nasal cavity occurs via the Ad fast responding mechanoreceptor pain fibers and slower unmyelinated C fibers. Activated fibers release tachykinins, including substance P, neurokinin A, and neuropeptide K.[34] Sympathetic stimulation is associated with neuropeptide Y, along with norepinephrine, and parasympathetic fibers cause release of acetylcholine and vasoactive intestinal peptide.[25,34,36,37].

Because the chemicals and neurotransmitters for paranasal nerve activation are the same as those found in migrainelike headaches, rhinogenic pain and allergic rhinitis often mimic each other, and therein is the conundrum.[34,38–40] Stimulation of the paranasal trigeminal nerves may cause reflexive changes in the body when afferent signals go to the trigeminal medullary sensory nucleus, where internuncial neurons in the reticular formation link to efferent parasympathetic vagus neurons in the motor nucleus with resultant vagal symptoms, including cardiac bradyarrhythmia, gastric hypermotility, and hypotension.[41] Although most "rhinogenic headaches" are migraines, approximately 80% to 90% of the time, there are cases where paranasal sinus pathologic condition can be responsible for headaches, as illustrated in the following set of clinical cases.

### Clinical Cases

#### Sphenoid sinusitis

Sphenoid sinusitis is a unique entity seen in approximately 3% of all sinusitis cases, which if delayed or misdiagnosed can result in high morbidity and mortality.[10,42–44] Headache is often the most common symptom in sphenoid sinusitis, and clinical and endoscopic assessment may be limited for assessment of disease. Because only a thin bony margin separates the sphenoid sinus from adjacent meninges, cavernous sinus, including the internal carotid arteries, cranial nerves III, IV, V, and VI, clivus, and pons, these structures are at risk in patients with severe sphenoid sinus disease. Patients with sphenoid sinusitis can present with headache, worse on standing, bending, movement, or coughing, with periorbital pain, and greater than 50% of patients may have a fever.[10,42,45] Unfortunately, physical examination is limited, and this diagnosis is often delayed; a key clue is the presence of a continuous increasing headache, CN V pain and paresthesia, photophobia, and eye tearing, with the headache causing sleep interference not relieved by analgesics.[10,42] Concern for sphenoid sinus pathologic condition requires imaging as seen in **Fig. 5**A–I, either via computed tomography (CT) or MR imaging, and if concern for vascular involvement as

demonstrated in this case, additional imaging, including CT angiography and interventional cerebral angiogram, may be warranted. These cases may result in complex pathology and require collaborative skills of otolaryngology and neurosurgery.[44]

### Epistaxis

Although epistaxis and migraine headaches may occur together, the exact causes are not well elucidated; however, as noted above, epistaxis is one of the most common ENT emergencies and may occur along the anterior nasal septum from involvement of Little or Kisselbach plexus. Severe dangerous cases of the posterior septum may result in airway compromise.[46] Tumors within the nasal fossa may present with nonspecific findings, which can lead to a delay in diagnosis; therefore, clinicians should pay special attention to clinical signs of nasal obstruction with epistaxis because these signs are suggestive of an underlying mass[47] (**Fig. 6**). Tumoral masses in the paranasal region, like all cancers in the head and neck, should prompt a careful radiographic assessment of the pterygopalatine fossa and ganglion, for loss of the fat planes, enlargement and enhancement of trigeminal and facial branches, including the vidian GSPN and deep petrosal nerves to evaluate for potential perineural tumoral involvement.[26]

In young male patients with epistaxis, nasal obstruction, and headache, a juvenile nasopharyngeal angiofibroma needs to be excluded.[48] These rare highly vascular tumors typically involve male adolescents and may also track along the skull base foramina intraorbitally or intracranially, as in **Fig. 7**.

Benign lesions may also obstruct the nasal cavities; interestingly, obstruction in and of itself may not necessarily cause pain or headache because the sinuses may be insensitive to pain.[10,49] The pain experienced in the sinus region may be a reflection of autonomic system engorgement with inflamed nasal structures along the nasofrontal ducts, turbinates, ostia, and upper nasal areas. True sinus headaches are reported to be more of a dull deep aching quality, with heaviness and fullness that does not present with nausea or vomiting.[10,49] The last important clinical case of epistaxis, shown in **Fig. 8**, is of an inverting papilloma or Schneiderian papilloma inverted type.

These locally aggressive benign lesions may recur and can be associated with carcinoma.[50] These are usually in male patients 50 to 70 years of age, with tumor arising from the lateral nasal wall of the ostiomeatal complex of the middle meatus often involving the maxillary sinus, with
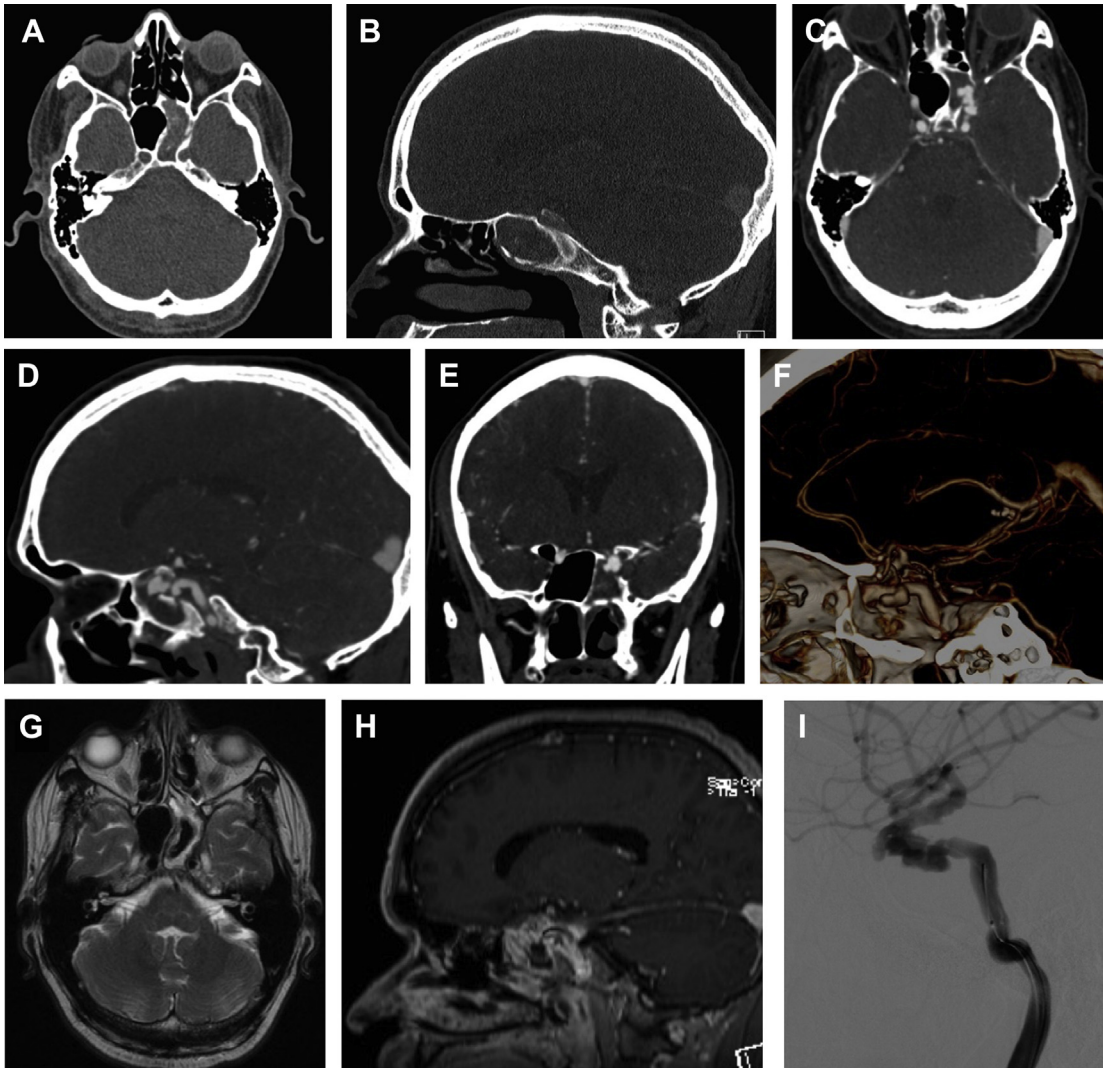
**Fig. 5.** (*A*) CT scan of the paranasal sinuses in the (*A*) axial plane, (*B*) sagittal plane of a geriatric female patient transferred from a nursing home, with a history of diabetes complaining of headache, demonstrating a left sphenoid sinus mucocele with foci of increased attenuation likely inspissated secretions or fungal infection in patient with aspergillus infection and dehiscence left lateral sphenoid bony margin, concerning for cavernous sinus and carotid artery involvement. (*C–F*) CT angiogram (*C*) axial plane, (*D*) sagittal plane, (*E*) coronal plane, and (*F*) 3-dimensional CT angiogram reconstruction showing multilobulated pseudoaneurysm of the left internal cavernous carotid artery. (*G*) Axial T2 MR imaging. Note lack of T2 signal in the left sphenoid sinus, from inspissated secretions with lack of mobile free water hydrogens making the sinus appear dark and aerated compared with (*H*) sagittal T1 postcontrast MR imaging demonstrating the enhancing mucosal margins, opacified left sphenoid mucocele, and multilobulated left internal carotid artery pseudoaneurysm projecting into the left sphenoid sinus. (*I*) Cerebral angiography of the left internal cavernous carotid artery multilobulated pseudoaneurysm with catheter in situ. The pseudoaneurysm and left cavernous ICA were coiled, and the patient had good collateral flow to the left middle cerebral artery via patient anterior and posterior communicating arteries.

unilateral nasal obstruction and epistaxis as the most common presenting symptoms. Causes are unknown; however, research has suggested that human papillomavirus (HPV) may be associated. HPV is a well-established cause for oropharyngeal carcinoma; of note, the sinonasal cavity is emerging as an additional critical area for transcriptionally active HPV-related tumors, a factor that should be taken into consideration when evaluating patients with recurrent nasal obstruction and headache with or without epistaxis.[51–54]
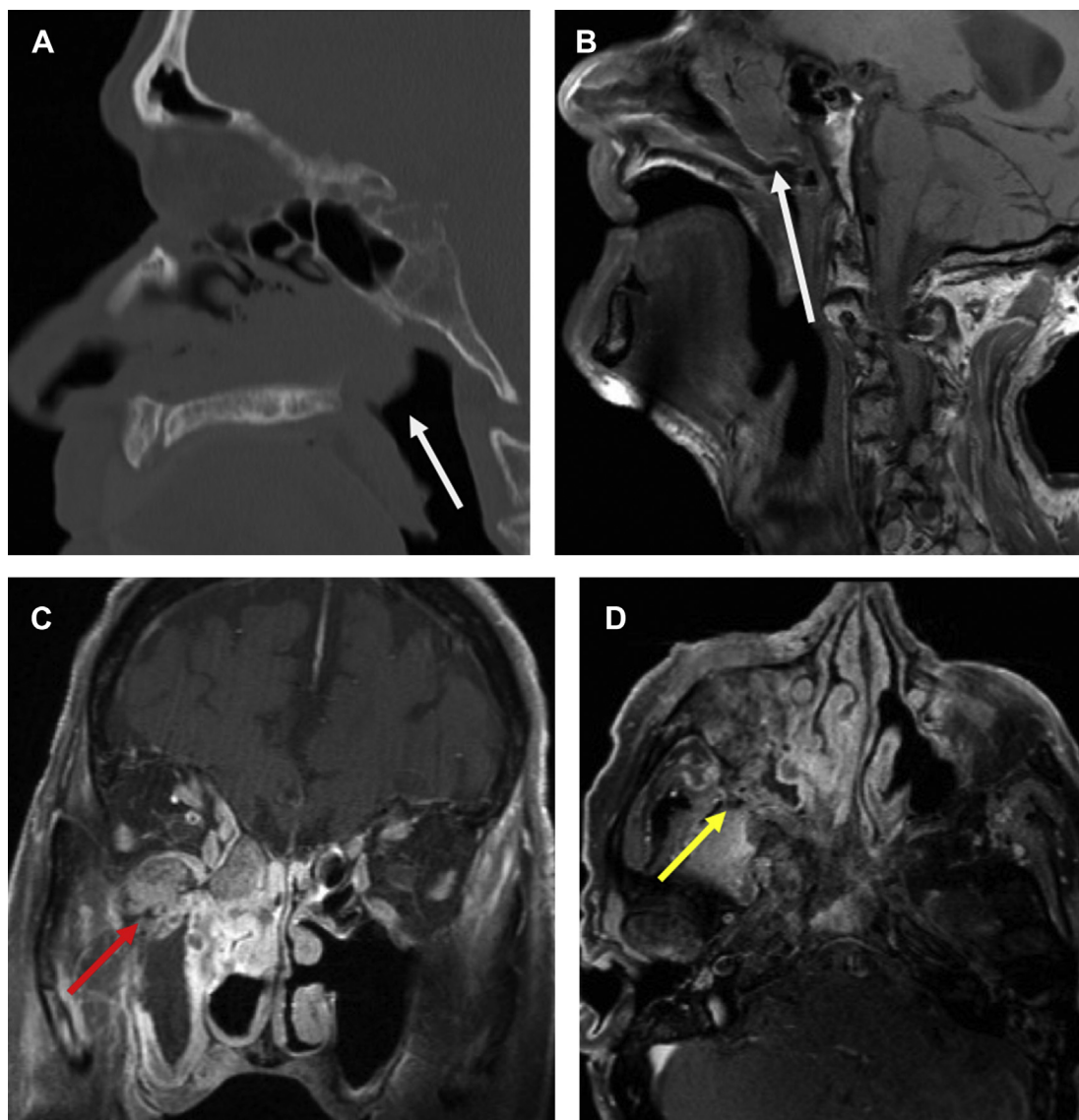
**Fig. 6.** (*A*) Sagittal CT, (*B*) sagittal T1 MR imaging in an 83-year-old man with nasal obstruction and epistaxis, and tumoral mass obstructing the right nasal cavity (*white arrows*), determined to be squamous cell carcinoma. Patient underwent surgical resection and radiation. (*C*, *D*) Coronal and axial T1 postcontrast gadolinium MR imaging with fat saturation, obtained 6 months later; patient with recurrent nasal obstruction, tumor, posterior epistaxis, and headache. (*C*) Perineural involvement (*red arrow*) along the right V$_2$ maxillary nerve in the infraorbital foramen lifting the right inferior rectus muscle superiorly. (*D*) Involvement of the right pterygopalatine fossa and pterygomaxillary fissure (*yellow arrow*). Perineural tumor involvement was also noted on the vidian nerve (GSPN) and right geniculate ganglion.

## How to Image

### Computed tomography

CT is a primary modality of choice for imaging the paranasal sinuses and assessment of the bony architecture and osseous margins. However, like all ionizing radiation imaging modalities, care must be taken to reduce unnecessary radiation exposure to radiosensitive organs, including the thyroid gland or orbit. CT imaging may be acquired using noncontrast, high-resolution, thin-section axial images reconstructed in the coronal and sagittal plane using both soft tissue and bone window algorithms. Contrast may be given in cases whereby there is concern for complications, including subperiosteal abscess, epidural or subdural empyema, osteomyelitis, tumor,
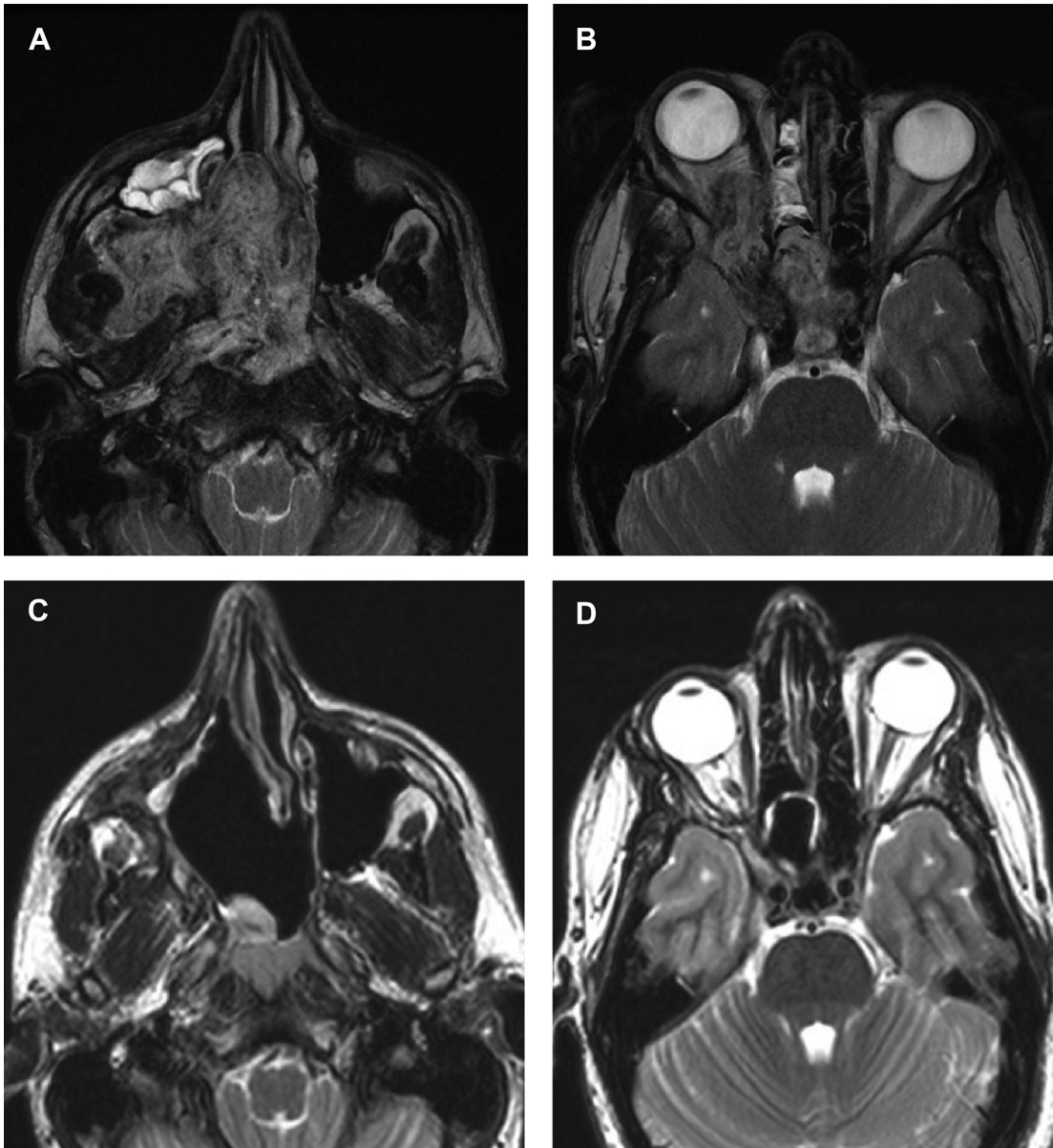
**Fig. 7.** (*A*, *B*) Axial T2 in a 17-year-old man with nasal obstruction and epistaxis, with hypervascular mass widening the pterygopalatine fossa, displacing the posterior maxillary sinus margin anteriorly with intraorbital and intracranial extension and right orbital exophthalmos. Patient underwent embolization via cerebral angiography and surgical resection. (*C*, *D*) Posttreatment axial T2, in the same planes, with removal of the mass, median antrectomy, turbinectomy and septectomy, and postoperative right orbital enophthalmos.

venous thrombosis, or concern for vessel involvement with additional imaging, including CT angiography or venography, as shown in **Fig. 5**. Iodinated contrast is used for contrast in CT; because of its increased electron density and higher atomic number (Z = 53), it has a radiodensity of approximately 25 to 30 HU/mg mL with tube voltages of 100 to 120 kVp.[1,15,55–58]

### MR imaging

MR imaging offers a better assessment of soft tissue characteristics, extent of tumor, and/or infectious or inflammatory processes, including perineural spread, as seen in **Fig. 6**, and for assessing intraorbital or intracranial extension. Sequences used include high-resolution (3 mm) T1- and T2-weighted images of the sinonasal cavities with inclusion of the orbit, skull base, and
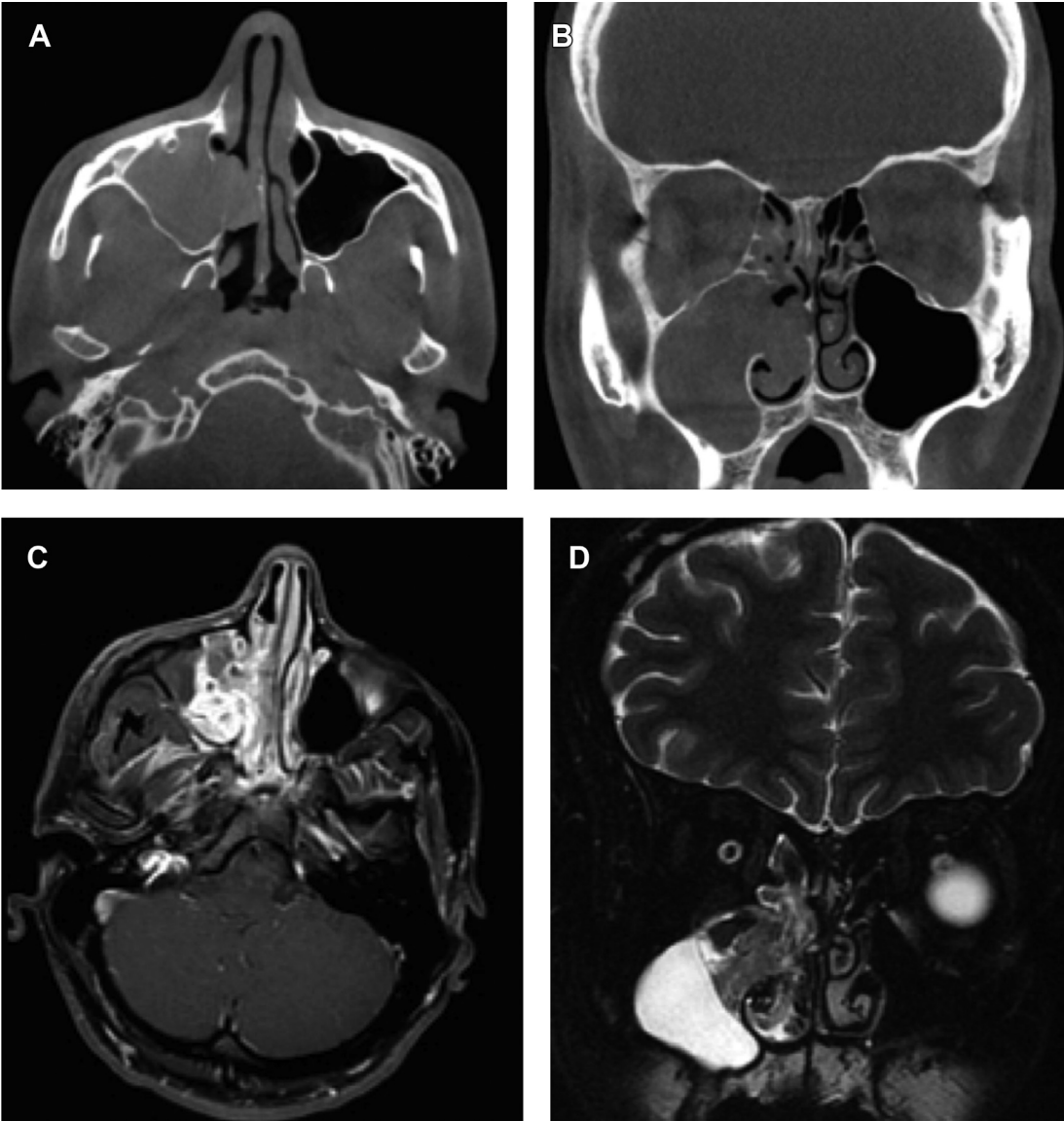
**Fig. 8.** (*A–D*) Bone window sinus CT axial and coronal (*A*, *B*) and MR imaging with gadolinium axial T1 with fat saturation (*C*) and coronal short TI inversion recovery (STIR) MR imaging (*D*), with a right inverting papilloma obstructing the right ostiomeatal complex with right maxillary mucocele. (*D*) The coronal MR imaging STIR sequence is able to separate the soft tissue component from the trapped bright secretions seen laterally, as opposed to the corresponding coronal sinus CT in (*B*).

adjacent intracranial regions; gadolinium contrast, which is paramagnetic because of 7 unpaired electrons, may be used. This shortens T1 and causes areas of increased vascularity or extravasation, as seen in **Figs. 5–7**, to appear bright on T1-weighted sequences.[1,15,57,58]

### Essential Clinical Findings

Although updated criteria for rhinosinusitis still include "pain" as an indicator for sinusitis,

research has shown a poor correlation between facial pain and headache and sinus disease, with many cases of self-diagnosed or referred "rhinosinusitis" actually being migraines.[5–8,59,60] Current diagnostic criteria for chronic rhinosinusitis include 12 weeks of nasal obstruction, congestion, anterior/posterior nasal discharge, facial pain/fullness, and decreased sense of smell, with objective verification of mucosal inflammation, polyps, or purulent discharge via CT or nasal endoscopy.[59,60] In fact, when

headache and facial pain are eliminated as one of the symptom-based criteria for chronic rhinosinusitis, the specificity of clinically and radiographically diagnosed sinusitis improves.[59,60] However, not all "rhinogenic headaches" are always migraines; in patients with a dull ache or epistaxis, or patients at risk for complications from sinusitis, imaging is vital for assessment of involvement of deep tissue planes, intraorbital or intracranial extension.

## SUMMARY

Headaches and sinus disease are common complaints with overlapping symptoms secondary to the autonomic innervation, with a marked worldwide prevalence. Most self-diagnosed "rhinogenic headaches" are actually migraines. However, certain headaches can signal sinus pathologic condition, and clinical history is important, especially in patients with dull, unrelenting positional headaches that may signal sphenoid sinus involvement. Awareness of the critical bony, vascular, and neural anatomy, clinical symptoms, and clinical history is vital. In patients with obstruction, nasal epistaxis, severe infectious or inflammatory pathologic condition, imaging is critical to assess vascular, orbital, or intracranial involvement, and treatment may require coordinated team involvement of Radiology, Otolaryngology, Neurology, and Neurosurgery.

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